L. schreibersi and from 26 to 72 mm ($\bar{x}$ = 49.6) in *L. barahonensis*. Both males and females were infected, and reproductive condition was insignificant (Williams' corrected *G*-test, $P < 0.05$). Juveniles of both sexes, reproductively active males, and females with unyolked and yolked ovarian follicles, oviducal eggs, and corpora lutea were infected. Habitat (i.e., access to water) did not appear important (Williams' corrected *G*-test, $P < 0.05$).

Stomachs of *Leiocephalus semilineatus* Dunn, 1920 (N = 35), and lizards in the genera *Anolis* (N = 146) (Iguanidae), *Ameiva* (N = 46) (Teiidae), *Hemidactylus* (N = 26) (Gekkonidae), and *Celestus* (N = 9) (Anguidae), taken from the same localities as infected specimens, were also examined. No physalopterid nematodes were found. Voucher specimens of *S. leiocephalorum* were deposited in the USNM Helminthological Collection (80581–80584) and the parasitological collection at Avila College, Kansas City, Missouri, U.S.A. (no numbers assigned).

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Research Note

Reduction of the *Syphacia* sp. Infection in the Laboratory Rat by Viprostol Treatment

MICHAEL R. IRWIN, REYNALDO J. ARCEO, AND THOMAS DAVIS

Experimental Pathology Department, Medical Research Division, American Cyanamid Company, Pearl River, New York 10965

ABSTRACT: During the conduct of routine chronic preclinical safety evaluation studies it was found that orally administered viprostol, a prostaglandin E_2 analogue, reduced or removed the *Syphacia* sp. infection in laboratory rats. This apparent anti-nematodal activity tended to correlate with the presence of gastrointestinal trophic changes, suggesting that the activity may be due to an altered environment of the parasite.

KEY WORDS: *Syphacia* sp., laboratory rat, prostaglandin E_2, viprostol.

We report an interesting observation of apparent anti-pinworm (*Syphacia* sp.) activity of a prostaglandin E_2 (PGE_2) analogue, viprostol, which was observed in 2 chronic preclinical toxicology studies conducted in rats (COBS CD.®, Charles River Breeding Laboratories Inc.). In the studies, groups of rats, unrestricted but individually housed, were treated with 0, 0.5, 2, or 10 mg/kg/day given orally in one study and applied topically in petrolatum in the other for 1 yr, followed by 1, 2, or 3 mo of posttreatment observation. Surviving rats were killed at the end of each period. A single stained tissue section of the colon of all rats was examined microscopically

Table 1. The prevalence of *Syphacia* sp. observed in rats after a year of viprostol treatment with or without a subsequent observation period.

Route	Dose mg/kg/day	Males				Females			
		After treatment period		After observation period		After treatment period		After observation period	
		N	% infected	*N*	% infected	*N*	% infected	*N*	% infected
Topical	0.00	25	20	30	43	25	16	29	17
	0.50	25	28	30	23	27	15	28	21
	2.00	27	0	28	14	27	0	28	11
	10.00	25	0	30	13	26	0	29	7
Oral	0.00	26	31	34	18	33	6	27	15
	0.50	28	0*	32	31	27	4	33	9
	2.00	27	0*	33	15	26	0	34	3
	10.00	33	0*	27	22	29	0	31	6

* $P < 0.05$.

as part of the routine histopathologic evaluation. The *Syphacia* sp., most probably *S. muris,* was identified (we thank Dr. Jack D. Tiner) using specimens dissected from formalin-fixed tissue. The criterion for establishing the infection in the various treatment groups was the presence of pinworm sections in stained (hematoxylin and eosin) colon tissue slides from individual rats.

Results of the evaluation are presented in Table 1. In both studies, no pinworms were observed in the colons of rats of both sexes treated orally or topically with either 2 or 10 mg/kg/day, nor in the males treated with 0.5 mg/kg/day orally. Using Fisher's exact test, the males treated orally with viprostol were the only ones to show statistical significance ($P < 0.05$). The female rats treated with 0.5 mg/kg/day had a prevalence of pinworms similar to the vehicle-treated controls. The rats treated with 2 or 10 mg/kg/day of viprostol apparently lost the pinworm infection and remained free of it while treatment was being administered. During the recovery period following treatment for 1 yr, the previously treated rats became rapidly reinfected within a month following the cessation of treatment.

Investigators using the *Nippostrongylus brasiliensis* rat model have suggested that the mechanism of PGE worm expulsion from the small intestine may either be by the direct effect on worm metabolism or a change in the gastrointestinal environment (Richards et al., 1977). We believe that the latter mechanism may be involved with *Syphacia* sp., as the prevention of epithelial gastrointestinal cell exfoliation, thought to be responsible for the PGE_2 cytoprotection (Reinhart et al., 1983), was observed in both orally and topically PGE-treated rats in these studies. These trophic gastrointestinal epithelial changes were not found in rats after a treatment-free period of 1 mo, during which the infection reappeared. No anthelmintic activity was observed in a standard diet test using 200 ppm of viprostol on 7–11-day-old *Trichostrongylus colubriformis* in gerbils. Viprostol may therefore be added to the list of compounds, like cimetidine (Rew and Fetterer, 1986), having an indirect antinematodal activity by altering the environment of the parasite.

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